

**CIRCULATORY FILLING PRESSURES  
DURING TRANSIENT  
MICROGRAVITY INDUCED BY  
PARABOLIC FLIGHT**

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## BACKGROUND

Theoretical concepts hold that blood in the gravity-dependent portion of the body would relocate to more cephalad compartments under microgravity conditions [1]. The result is an increase in blood volume in the thoracic and cardiac chambers. This increase in central volume shift should result in an increase in central atrial filling pressures. However, experimental data has been somewhat contradictory and nonconclusive to date. Early investigations of peripheral venous pressure and estimates of central venous pressure (CVP) from these data did not show an increase in CVP in the microgravity condition [2,3]. However, CVP recorded in human volunteers during the parabolic flight by Norsk revealed an increase in CVP during the microgravity state [4]. On the June 1991 STS 40 shuttle mission, a payload specialist wore a fluid line that recorded CVP during the first few hours of orbital insertion. These data revealed decreased CVP. When this CVP catheter was tested during parabolic flight in four subjects, two subjects had increased CVP recordings and two other subjects had decreased CVP measurements. In April 1991, our laboratory performed parabolic flight studies in several chronic-instrumented baboon subjects. It was again noted that centrally recorded right atrial pressure varied with exposure to microgravity, some animals having an increase and others having a decrease. Thus, data presently available has demonstrated a variable response in the mechanism not clearly defined. In April 1992, we determined a test hypothesis relating the possible mechanism of these variable pressure responses to venous pressure-volume relationships.

## METHODS

Our study used mature male baboons, 20-25 kg. Animals were first acclimated to the study environment that included a custom-designed confinement chair and instrumentation jackets to protect exteriorized leads. Surgical implantation was performed under general anesthesia and sterile conditions via a left thoracotomy approach as previously described [5]. Custom-designed doppler dimension transducers were placed in the ascending aorta pulmonary artery. Pressures were measured by Konigsberg pressure cells adjacent to the flow transducers. A pressure cell was also placed in the

right ventricular apex. Custom-designed ventricular access ports were placed in the left ventricular apex, and right and left atria. The animals were allowed to recover for two weeks and then underwent catheterization to calibrate the transducers.

Parabolic flight profiles used NASA's modified KC-135, operated by the Johnson Space Center Reduced Gravity Program. The aircraft was flown from Ellington Field, Houston, Texas, and staged from Kelly Air Force Base, San Antonio, Texas, near LACR facilities. Five days of flights were performed. Each flight day included 40 parabola flown in 4 sets of 10. The set of 40 parabola was preceded and concluded with the push-over maneuver to avoid the near 2g acceleration with the pull-up. Two animals per day were flown for hemodynamic studies and one sedated and intubated animal was flown to perform transesophageal echo. One of the hemodynamic animals had been pretreated with furosemide the night prior to study to result in a volume-contracted state, and the second animal was given 500 cc fluid bolus just prior to flight. All data was recorded on VHS analog recorders. Data was post-processed by playback of atrial pressure data on a Gould ES 1000 strip chart recorder.

## RESULTS

Mean right atrial pressures (RAP) during 1991 flights had a variable early microgravity response: increases in  $n=3$  and decreases in  $n=3$  (supine) and increases in  $n=5$ , decreases in  $n=2$  (upright). In 1992 flights, the RAP change in volume-depleted (VD) subjects from pull-up to microgravity was not significantly different between upright ( $-10 \pm 4.1$  mmHG) and supine positions ( $-3.2 \pm 2.2$  mmHG,  $p>.05$ ). In contrast, RAP change in volume-expanded (VE) subjects was significantly ( $p<.01$ ) greater upright,  $13.1 \pm 1.5$  mmHG, than supine,  $4.2 \pm 2.8$  mmHG. VD values were significantly different from VE ( $p<.01$ ) for supine and upright postures. The results are shown in Figure 1. Euvoletic (EU) increased with microgravity  $+6.9 \pm .9$  mmHg (upright only). Left atrial pressure (LAP) responses were similar, but more variable.

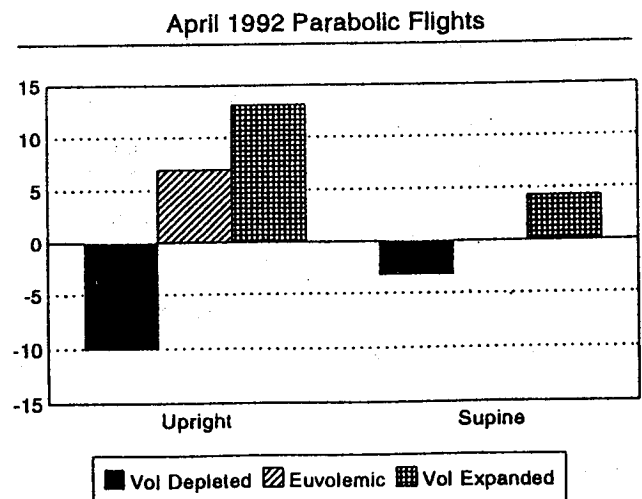


Figure 1. RAP changes from pull-up to microgravity

The LAP responses were more variable. VD animals showed an increase of  $+5.9 \pm 3.6$  mmHg upright compared to a decrease of  $-5.0 \pm 1.3$  mmHg supine. EU demonstrated an average increase of  $+9.0 \pm 0.8$  mmHg upright (no supine data). Finally, LAP in the VE group increased  $17 \pm 1.8$  mmHg upright compared to  $6 \pm 1.4$  mmHg supine. Pressure changes in the LA lagged those of the RA by several beats. Average from 4 baboons is shown in Figure 2.

April 1992 Parabolic Flights

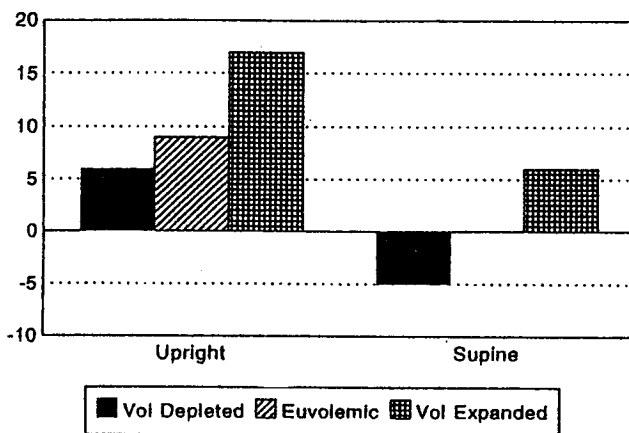


Figure 2. LAP changes from pull-up to microgravity

## DISCUSSION

Our data revealed that central filling pressures are significantly determined by the initial volume status of the subject and also somewhat dependent upon posture. When animal core volume depleted, the response to microgravity for CVP is a decrease. CVP increases in euvolemic and volume-expanded animals. Theoretically, we presume this is because in the upright posture the venous compartments out of the chest and above the heart level are essentially collapsed. Some may become expanded in a microgravity environment and contribute to the overall capacitative effects of the venous system. In a volume-depleted status this is enough to result in a decrease in the overall pressures response. When the animal is supine, veins in the legs, thighs and calves, contribute to the potential capacitative compartment that becomes a contributor in the microgravity state. The volume-expanded state allows for enough fluid to shift to fill this compartment and result in a pressure increase instead of a decrease. The left atrial pressure presumably lags behind the right atrial pressure changes due to transmission time through the lungs.

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